REMARKS

Claims 1-45 were presented at the time of filing and are currently pending in the application with claims 15-30 withdrawn from consideration. Claims 15-30 are canceled above. Claims 1-14 and 31-45, therefore, remain pending in the application.

Drawings

The Office Action includes an objection to the drawings, due to the appearance of the attorney docket number at the top of each drawing. Submitted herewith are replacement sheets for Figures 1-13 in which the attorney docket number has been replaced with the application serial number.

In light of the above amendment, Applicant respectfully requests the withdrawal of Examiner's objections to the drawings.

Rejections under 35 U.S.C.§112, first paragraph

Claims 1-14 and 31-45 are rejected under 35 U.S.C.§112, first paragraph, as failing to comply with the written description requirement, that is, that the claims contain subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor, at the time the application was filed, had possession of the claimed invention. Applicant respectfully but strenuously disagrees.

The claims are directed to a method for assessing the potential of a compound to function as an anti-arrhythmic agent using a cell that expresses a recombinant mutant Nav 1 sodium channel protein. The claim recites a mutant sodium channel protein having an amino acid sequence in which one or more amino acids among the ten amino acids occurring at the carboxy end of the S6 segments of D1, D2, D3 or D4 domains of a mammalian Nav1 protein *differs from the amino acid in wild-type Nav1* by substitution with tryptophan, phenylalanine, tyrosine or

cysteine. In other words, the mutant sodium channel protein of the invention has the same amino acid sequence as the corresponding wild-type mammalian Nav 1, **except** that one or more amino acids among the ten amino acids occurring at the carboxy end of the S6 segments of D1, D2, D3 or D4 domains of the mutant protein differs from the amino acid in wild-type Nav1 by substitution with one of tryptophan, phenylalanine, tyrosine or cysteine. Substitution with any other amino acid residue in any other portion of the molecule is not contemplated by or claimed in the instant application.

Surprisingly, the Office Action concludes that the claims do not require that the polypeptides possess any particular conserved structure or any other disclosed distinguished feature and alleges that the claims, therefore, are drawn to a genus of that is defined by a large number of amino acid substitutions which the Office Action characterizes as "innumerable possibilities."

In fact, the mutant sodium channel protein of the invention has the same overall sequence as the corresponding wild type molecule differing from the wild-type only by one or more amino acid substitutions in a very limited 10 amino acid region. Therefore, the number of possible mutants is much more limited than the Office Action would suggest.

Nav proteins consist of one large α-subunit which contains four homologous repeated domains (D1-D4) each with six transmembrane segments (S1-S6) (see Figure 1). The complete amino acid sequences for several isoforms of mouse, rat and human Nav 1 proteins are known and available in the NCBI database. The amino acid sequences of the four S6 regions of several rat and human isoforms is provided in the specification on pages 3 and 4. As disclosed in the specification on page 3, paragraph [0006], there is very close homology among the S6 segments of mammalian Nav proteins so far identified and that this homology extends both through species and isoforms. Applicant has identified the highly conserved carboxy terminal 10 amino acid region as being crucial to activation. Thus, one of skill in the art would expect that substitution in this highly conserved region will have the same effect on sodium channel function across

mammalian species and isoforms of the Nav 1 protein.

As discussed above, the substitutions encompassed by the present invention are limited to a region of ten amino acids occurring at the carboxy end of an S6 segment of each of D1, D2, D3 or D4. Furthermore, these specific 10 amino acid positions can only be substituted with one of four possible replacement amino acids: tryptophan, phenylalanine, tyrosine or cysteine. The claimed invention does not therefore, as the office action alleges, encompass other mutations, substitutions, insertions or deletions in other portions of Nav 1.1, Nav 1.2...Nav 1.9. In actuality, the number of possible mutant sodium channels contemplated by the invention are much more limited than the office action would suggest.

The Office Action cites University of California v. Eli Lilly as being applicable to the present case for the proposition that Applicant's claims encompass a very large undisclosed genus. Applicant respectfully disagrees. A description of a genus may be achieved by means of a recitation of a representative number of species falling within the scope of the genus or of a recitation of structural features common to the members of the genus, which features constitute a substantial portion of the genus. In Lilly, it was held that the written description requirement as applied to certain biotechnology patents, in which a gene material has been defined only by a statement of function or result, is not satisfied by such a statement. In Lilly, the court concluded that a claim to a microorganism containing a human insulin cDNA was not adequately described by a description of rat insulin cDNA and a statement that the invention included human insulin. In Lilly, the nucleic acid sequences for insulin of other species including human had not yet been identified.

Unlike the present case, the term human insulin cDNA in Lilly's claim conveyed no distinguishing information about the identity of the claimed DNA sequence, such as its relevant structural or physical characteristics. An adequate written description of genetic material, according to the court, requires "a precise definition, such as by structure, formula, chemical name, or physical properties, not a mere wish or plan for obtaining the claimed chemical

invention." The specification in the Lilly case therefore did not demonstrate that the inventors had possession of human insulin cDNA.

Unlike Lilly, where only one species was disclosed with no additional information regarding homology of the disclosed sequence with others of the claimed genus, Applicant discloses two mammalian species, rat and human, and multiple isoforms for each species. Additionally, the specification includes comparison of the amino acid sequences for the relevant region, i.e. the S6 transmembrane segments of D1, D2, D3 and D4 where the mutations are to be made. With minor exception, the rat and human Nav 1 sequences are identical.

Additional amino acid sequences for Nav 1proteins in the mouse can also be found in the NCBI database, for example, accession no. Q9WTU3, Nav 1.6 in mouse (a copy is enclosed herewith for the Examiner's convenience.) Because of the very high degree of homology (and in some cases complete identity) of the 11 amino acids at the carboxy terminal of the S6 segments, the person of skill in the art would expect that substitution in this region will have the same effect on sodium channel function across mammalian species and isoforms of the Nav 1 protein. By extension, based on the written description of the invention in the specification, one of skill would recognize that Applicant was in possession of the invention at the time the application was filed.

The Office Action also cites Fiers v. Revel, Amgen v. Chugai Pharmaceutical and Fiddes v. Baird for the proposition that "the skilled artisan cannot envision the detailed chemical structure of the encompassed genus of polypeptides, and therefore conception is not achieved until reduction to practice has occurred..." These cases are completely inapposite to the instant case. As already discussed, the complete amino acid sequences are known for several Nav 1 isoforms for mouse, rat and human. Based on Applicant's research, the mutations contemplated by the present invention occur in a specific, well elucidated region. Thus the skilled artisan can easily discern what the chemical structure of the mutant sodium channel is.

In the instant case, Applicant has adequately described the mutant sodium channels encompassed by the invention by providing the precise structure/sequence of the relevant regions of the wild type molecule and further delineated the molecule by specifically defining the amino acid positions to be substituted, i.e., the ten amino acids occurring at the carboxy end of the S6 segments of D1, D2, D3 an D4. Furthermore, replacement of amino acids in the pertinent 10 amino acids are limited to substitution with either tryptophan, phenylalanine, tyrosine or cysteine. Applicant respectfully submits that it is unclear how one could provide a more precise definition. Clearly, from this description, the skilled artisan could, in fact, envision the detailed chemical structure of the claimed genus of polypeptides.

Therefore, in view of the fact that Applicant has identified a specific structural region of known Nav 1 proteins believed to be involved in channel activation, and has been able to modify channel activation by making very limited amino acid replacements in this region, it is believed that the specific examples of cells containing a mutant sodium channel protein provided in the specification clearly provide an adequate written description of the invention.

In view of the above remarks, the rejection under 35 U.S.C. §112, first paragraph of claims 1-14 and 31-45 based on lack of written description is believed overcome. Withdrawal of the rejection is respectfully requested.

It is respectfully submitted that the above-identified application is now in condition for allowance and favorable reconsideration and prompt allowance of these claims are respectfully requested. Should the Examiner believe that anything further is desirable in order to place the application in condition for allowance, the Examiner is invited to contact Applicants' undersigned attorney at the telephone number listed below.

Respectfully submitted,

Kathy Smith Dias

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Dated: August 5, 2005

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Amendments to the Drawings:

The attached sheets of drawings (each labeled "Replacement Sheet") contain Figures 1-13.

Attachment: Replacement Drawing Sheets

1: Q9WTU3. Reports Sodium channel pr...[gi:34098761]

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LOCUS       Q9WTU3                  1978 aa            linear   ROD 13-SEP-2005
DEFINITION  Sodium channel protein type VIII alpha subunit (Voltage-gated
            sodium channel alpha subunit Nav1.6).
ACCESSION   Q9WTU3
VERSION     Q9WTU3  GI:34098761
DBSOURCE    swissprot: locus SCN8A_MOUSE, accession Q9WTU3;
            class: standard.
            extra accessions:Q60828,Q60858,Q62449,created: Oct 10, 2003.
            sequence updated: Oct 10, 2003.
            annotation updated: Sep 13, 2005.
            xrefs: AF049617.1, AAD20438.1, U26707.1, AAC52242.1, U59964.1,
            AAC52708.1, U59963.1, U23158.1, AAA65599.1
            xrefs (non-sequence databases): HSSPP04775,
            EnsemblENSMUSG00000023033, MGI103169, GO0001518, GO0005248,
            GO0007628, GO0007626, GO0007517, GO0006814, InterProIPR001682,
            InterProIPR002111, InterProIPR005821, InterProIPR000048,
            InterProIPR005820, InterProIPR001696, InterProIPR008054,
            InterProIPR010526, PfamPF00520, PfamPF00612, PfamPF06512,
            PRINTSPR00170, PRINTSPR01667, PROSITEPS50096
KEYWORDS    Alternative splicing; ATP-binding; Disease mutation; Glycoprotein;
            Ion transport; Ionic channel; Multigene family; Nucleotide-binding;
            Polymorphism; Repeat; Sodium; Sodium channel; Sodium transport;
            Transmembrane; Transport; Voltage-gated channel.
SOURCE      Mus musculus (house mouse)
  ORGANISM  Mus musculus
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia;
            Sciurognathi; Muroidea; Muridae; Murinae; Mus.
REFERENCE   1  (residues 1 to 1978)
  AUTHORS   Plummer,N.W., Galt,J., Jones,J.M., Burgess,D.L., Sprunger,L.K.,
            Kohrman,D.C. and Meisler,M.H.
  TITLE     Exon organization, coding sequence, physical mapping, and
            polymorphic intragenic markers for the human neuronal sodium
            channel gene SCN8A
  JOURNAL   Genomics 54 (2), 287-296 (1998)
   PUBMED   9828131
  REMARK    NUCLEOTIDE SEQUENCE (ISOFORMS 1 AND 3).
            STRAIN=C57BL/6J
REFERENCE   2  (residues 1 to 1978)
  AUTHORS   Burgess,D.L., Kohrman,D.C., Galt,J., Plummer,N.W., Jones,J.M.,
            Spear,B. and Meisler,M.H.
  TITLE     Mutation of a new sodium channel gene, Scn8a, in the mouse mutant
            'motor endplate disease'
  JOURNAL   Nat. Genet. 10 (4), 461-465 (1995)
   PUBMED   7670495
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REMARK NUCLEOTIDE SEQUENCE (ISOFORM 2), TISSUE SPECIFICITY, AND DISEASE.
STRAIN=C57BL/6J; TISSUE=Brain
REFERENCE 3 (residues 1 to 1978)
AUTHORS Kohrman,D.C., Harris,J.B. and Meisler,M.H.
TITLE Mutation detection in the med and medJ alleles of the sodium channel Scn8a. Unusual splicing due to a minor class AT-AC intron
JOURNAL J. Biol. Chem. 271 (29), 17576-17581 (1996)
PUBMED 8663325
REMARK NUCLEOTIDE SEQUENCE OF 93-205, AND DISEASE.
STRAIN=129/Sv; TISSUE=Brain
REFERENCE 4 (residues 1 to 1978)
AUTHORS Fan,Z., Kyle,J.W. and Makielski,J.C.
TITLE Direct Submission
JOURNAL Submitted (??-MAR-1995)
REMARK NUCLEOTIDE SEQUENCE OF 1411-1686.
REFERENCE 5 (residues 1 to 1978)
AUTHORS Plummer,N.W., McBurney,M.W. and Meisler,M.H.
TITLE Alternative splicing of the sodium channel SCN8A predicts a truncated two-domain protein in fetal brain and non-neuronal cells
JOURNAL J. Biol. Chem. 272 (38), 24008-24015 (1997)
PUBMED 9295353
REMARK ALTERNATIVE SPLICING (ISOFORMS 1; 4 AND 5).
TISSUE=Brain, and Fetal brain
REFERENCE 6 (residues 1 to 1978)
AUTHORS Kohrman,D.C., Smith,M.R., Goldin,A.L., Harris,J. and Meisler,M.H.
TITLE A missense mutation in the sodium channel Scn8a is responsible for cerebellar ataxia in the mouse mutant jolting
JOURNAL J. Neurosci. 16 (19), 5993-5999 (1996)
PUBMED 8815882
REMARK VARIANT MEDJO THR-1317, AND VARIANT LEU-5.
STRAIN=DBA/2WyDi
REFERENCE 7 (residues 1 to 1978)
AUTHORS De Repentigny,Y., Cote,P.D., Pool,M., Bernier,G., Girard,S., Vidal,S.M. and Kothary,R.
TITLE Pathological and genetic analysis of the degenerating muscle (dmu) mouse: a new allele of Scn8a
JOURNAL Hum. Mol. Genet. 10 (17), 1819-1827 (2001)
PUBMED 11532991
REMARK DISEASE.
COMMENT [FUNCTION] Mediates the voltage-dependent sodium ion permeability of excitable membranes. Assuming opened or closed conformations in response to the voltage difference across the membrane, the protein forms a sodium-selective channel through which Na(+) ions may pass in accordance with their electrochemical gradient.
[SUBCELLULAR LOCATION] Integral membrane protein.
[ALTERNATIVE PRODUCTS] Event=Alternative splicing; Named isoforms=5; Name=1; Synonyms=18A; IsoId=Q9WTU3-1; Sequence=Displayed; Name=2; IsoId=Q9WTU3-2; Sequence=VSP_050594; Name=3; IsoId=Q9WTU3-3; Sequence=VSP_050595; Name=4; Synonyms=18N; IsoId=Q9WTU3-4; Sequence=VSP_050596, VSP_050597; Name=5; IsoId=Q9WTU3-5; Sequence=VSP_050598.
[TISSUE SPECIFICITY] Expressed in brain, cerebellum and spinal cord.
[DOMAIN] The sequence contains 4 internal repeats, each with 5 hydrophobic segments (S1,S2,S3,S5,S6) and one positively charged segment (S4). Segments S4 are probably the voltage-sensors and are characterized by a series of positively charged amino acids at every third position.
[DISEASE] Defects in Scn8a are the cause of motor endplate disease (med). Med is a recessive neuromuscular disorder that is

characterized by lack of signal transmission at the neuromuscular junction, excess preterminal arborization and degeneration of cerebellar Purkinje cells. It produces early onset progressive paralysis of hind limbs, severe muscle atrophy and juvenile lethality.
[DISEASE] Defects in Scn8a are the cause of the jolting mutant (medjo), a mild form of motor endplate disease which is characterized by the absence of spontaneous, regular, simple discharges from Purkinje cells. After 3 weeks of age, jolting mice are unsteady and have wide-based gait and a rhythmical tremor of head and neck induced by attempted movement.
[DISEASE] Defects in Scn8a are a cause of degenerating muscle (dmu). Dmu is an autosomal recessive neuromuscular disorder that is characterized by skeletal and cardiac muscle degeneration. It produces early onset progressive loss of mobility of the hind limbs and subsequent lethality in the first month of life.
[SIMILARITY] Belongs to the sodium channel family.
[SIMILARITY] Contains 1 IQ domain.

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Protein	1..1978
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D2S6

D3S6

D4S6

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